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LABORATORY GUIDE

TO

PATHOLOGICAL HISTOLOGY

BY

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Technique.

The staining solutions most commonly employed are the following :—

I. Delafield's Haematoxylin. See "Mallory & Wright."

II. Ehrlich's Acid Haematoxylin. See "Mallory & Wright."

III. Methyl Blue.

IV. Saffranin.

V. Alum Carmine.

VI. Alcoholic Eosine as a counter-stain.

The following methods are employed in staining with the Ehrlich's Acid Haematoxylin solution:—

1. Stain for from three to five minutes.

2. Wash in tap water till tissue assumes blue color (about fifteen minutes), or if a few drops of 10 percent ammonia are added to the water the process is much shortened.

3. Wash in water.

4. Place in absolute alcohol to which a few drops of eosine have been added for one minute, tissue is thus dehydrated and counter-stained at same time.

5. Clear in xylol, turpentine and creosote, bergamot oil or any of the other clearing reagents.

6: Mount in Canada Balsam.

With tissues which have been kept in preserving fluids for a long period it is found very difficult to stain by the ordinary method so that they require to be over stained for from five to seven, or even ten minutes, then washed in 3 percent acetic acid solution, washed in water or ammoniated water and proceeded with in the ordinary way.

In using the alum stains the tissues must be well washed in water to remove the alum.

Methods employed to determine the different forms of degeneration:—

1. CLOUDY SWELLING :—If to a cloudy degenera-

tion a few drops of glacial acetic acid are added, the granules will clear up, the cells becoming quite normal in appearance.

II. FATTY DEGENERATION :—(a) If to a fatty degeneration acetic acid be added no change takes place. If now some fat solvent such as ether, alcohol or chloroform, be added, the cells will at once clear up, as in cloudy swelling. In order to demonstrate the presence of fat the two following differential fat stains are employed :—

(b) Osmic acid stain (1 or 2 percent solution), the tissues being counter-stained with saffranin. The fat stains brown or black while the rest of tissue stains red.

(c) Sudan III. and Haematoxylin :—
Stain for usual time in Ehrlich's Haematoxylin .
Wash in water.
Stain five minutes in Sudan III.
Wash in water or very weak alcohol.
Mount in Farrant's solution or in glycerine.
By this method the fat takes on a golden or salmon that while the rest of tissue is stained blue.

III. AMYLOID DEGENERATION :—

(a) Stain in weak solution of Iodine for 3 minutes.
Wash in water.
Mount and examine in water, glycerine or Farrant's solution.

By transmitted light, the amyloid areas stain light canary yellow, while the rest of the tissue is much darker. By reflected light the opposite effect is produced, viz : the non-amyloid areas take on the canary-yellow stain.

(b) Stain in Methyl-aniline violet for two to three minutes.

Wash in 1 percent hydrochloric acid solution.
Wash in water.
Mount in Farrant's solution.

By this method amyloid areas stain reddish purple, while healthy tissue takes a deep blue stain.

Note that with all the differential stains for the degenerations the tissues are mounted directly in water, glycerine or Farrant's solution.

Stains for "NERVOUS SYSTEM" will be described under that section.

INFLAMMATION.

ETIOLOGY :—Mechanical, chemical, electrical, thermal and bacterial irritants.

MICROS. EXAM :—The changes that take place microscopically are the following :—First, at the point of injury the arterioles momentarily contract. This is rapidly followed by a dilatation of all the vessels in the part, accompanied by an acceleration in the blood flow. The current gradually slows, till, in some cases, stasis occurs, and this may or may not end in thrombosis. During the slowing of the current the white corpuscles tend to range themselves at the periphery of the tubes, later to adhere to the walls of the vessels, and then, gradually to penetrate between the endothelial cells by throwing out pseudopodia, and finally to penetrate into the tissues about the blood vessels. The red corpuscles may also pass through the vessel walls by a process of diapedesis. Before, and during the passage through the vessel walls, of these cellular elements, there is an exudation of serum (rich in albumen and fibrin ferment) into the tissues, causing an oedema. With this process, there is a proliferation of the preexisting connective tissue cells (fibroblasts).

The serum may become reabsorbed and the leucocytes may return to the blood current unchanged through the lymph channels, some disintegrate and are then absorbed, while others are thrown off as an exudate on a mucous or serous membrane. The red blood cells disintegrate and are absorbed. Any of the above products of exudation may predominate, giving rise to serus, fibrinous, serofibrinous or sanguineous inflammations. Macroscopically these changes are associated with increased redness, heat and swelling, with possibly an exudation of serum, fibrin, blood or pus. Thus the circulation may be restored or the inflammations may terminate in :—

A. Resolution as described previously.

B. Organization or fibroid thickening. This takes place when the blood in the vessels has coagulated. Here there is hyperplasia of connective tissue to be described under healing wound. With chronic inflammation the same changes take place.

. Suppuration, especially when parts are infected by pyogenic organisms. See ulcer and abscess.

D. Gangrene due to intensity of virus and weakness of tissues, as seen in senile gangrene and endarteritis obliterans. The tissue necroses en masse.

The inflammations, therefore, can be classed under the following headings :—

I. Exudative inflammation already described.

II. Necrotic inflammation described under ulcer and abscess.

III. Productive inflammation : The products of inflammation are here represented entirely by the hyperplasia of connective tissue and the formation of granulation tissue (the latter described under wound healing by granulation). This variety is associated with long continued inflammations, gout, rheumatism, alcohol and the infective granulomata, e.g., syphilis, tuberculosis and actinomycosis.

HEALING WOUND.

In the simplest form, i.e., where there is no loss of substance, but where the wound is made with sharp instruments, and the edges of the incision are brought together at once, there is here slight exudation of inflammatory lymph between the edges of the wound, which keeps them in apposition. Few new vessels traverse this tissue, and the wound heals by first intention. If there is loss of substance, as when the wound is caused by bruising or by a blunt instrument, then there is no adhesion of the edges of the wound, but rather the gap is filled up by an exudation of fibrinous lymph. The cells of the capillary walls in the neighborhood of the wound divide, and by uniting with other cells similarly given off form solid loops, which, later on, become canalized, thus forming the new vessels. These vessels penetrate the exudation, the fibroblastic cells (small round and spindle-shaped), proliferate around them. As the process goes on, the new vessels intercommunicate from side to side. The fibroblastic cells gradually become transformed into adult fibrous tissue. This connective tissue later undergoes cicatricial contraction, occluding and destroying many of the vessels. These loops of new vessels with their envelope of small round cells form the granulations which are seen on the surface of a granulating wound. On the skin surface the epidermis is replaced by a simple layer of

squamous epithelial cells derived principally from the Malpighian layer. This epithelial growth which contains neither hair follicles nor glands, takes place from the periphery towards the centre. As cicatricial contraction goes on the edges of the wound are brought into closer apposition, and thus the scar becomes comparatively small as compared with the size of the original wound.

"ULCER" OF SKIN.

From the inflammation or injury superficial tissue is destroyed, the gap being filled up with fibrinous lymph. Because of the presence of bacteria with their toxines, the surrounding vessels dilate and leucocytes emigrate into the exudate causing disintegration of the fibrin, and there forms in its place, a mass of necrotic tissue or "scab." As in healing wound, the surrounding vessels bud out and are surrounded by fibroblasts, "granulations," and then by the same process, as in healing wound, repair takes place. If the process is chronic there is marked proliferation throughout of fibrous tissue, and at the edges of the ulcer, the epidermis has proliferated and dips deeply down into subcutaneous tissue in long finger-like processes.

"ABSCESS."

At the centre of infection in the deeper tissues, or viscera, the vessels become congested. Leucocytes emigrate through their walls in great numbers, forming clumps of small round cells, these, later proliferate and disintegrate forming pus cells. These, together with the serum and fibrin, may liquify, forming a mass of liquid debris in the centre (pus). In the periphery the congested vessels are surrounded by large numbers of fibroblasts which proliferate rapidly. New capillary loops with their fibroblastic envelopes, as in healing wound, are formed, leading eventually, to the formation of a fibrous capsule. The whole abscess may thus become organized, or the pus may burrow through to the surface, or there may be a deposit of lime salts into the contained broken down tissue.

"GANGRENE."

From blocking of a vessel by a thrombus as in endarteritis obliterans, the whole tissue supplied by that ves-

sel may undergo coagulation necrosis, as seen in other necrotic tissue. In the tissue surrounding this area the vessels become congested, and there is a pouring out of inflammatory exudate, thus forming a line of demarcation. The whole of the tissue which underwent coagulation necrosis sloughs off en masse, and the raw surface may then heal as an ordinary ulcer. In extensive burns or frost-bites the same process takes place.

ALIMENTARY TRACT.

LIVER.

"LIVER"—Cloudy Swelling.

PREPARATION :—(a) Cut with freezing microtome, (b) Mount in Farrant solution. To a second section, add Acetic acid, after mounting. Compare both specimens.

ETIOLOGY :—Continued high fevers, especially those of infectious diseases.

MICROSCOPICAL EXAM :—Cells swollen and granular (granules composed of minute albuminoid bodies). Specimen to which the acetic acid has been added becomes quite clear, as albuminoid granules are thus dissolved. With ordinary stained specimens note the swelling of hepatic cells with badly staining and obscure nuclei.

MACROSCOPIC EXAM :—Liver enlarged, swollen, edges rounded, surface smooth and pale colored, capsule opaque. On section, pale colored and cloudy (parboiled appearance).

"LIVER"—Fatty infiltration.

PREPARATION :—(a) Cut with freezing microtome, (b) Stain three minutes in Ehrlich's Haemotaxylin, (c) Wash in water, (d) Stain five minutes in Sudan III., (e) Wash in very weak alcohol, (f) Mount in Farrant solution.

ETIOLOGY :—Sometimes physiologic, over eating

and general obesity, anaemia and cachetic conditions, and certain poison, e.g. Antimony, etc.

MICROSCOPICAL EXAM :—Note great deposit of fat in peripheral cells of lobules in form of large droplets. When extreme the whole lobule becomes affected. Fat is stained pink or salmon color by Sudan III. This change takes place about the nucleus but not in the cell nuclei.

MACROSCOPICAL EXAM :—Liver enlarged, edges rounded, consistence doughy, color yellowish.

"LIVER"—Fatty Degeneration.

PREPARATION :—Same as Fatty infiltration.

ETIOLOGY :—Severe anaemias, infectious fevers and intoxications, phosphorous poisoning, etc. It follows upon cloudy swelling.

MICROSCOPIC EXAM :—Liver cells filled with small granules or droplets of fat and not large as in infiltration. This change takes place both in and about the nucleus of the cells.

MACROSCOPIC EXAM :—Organ smaller and softer than normal, capsule frequently shrunken, color yellowish, substance friable, fat can be scraped from cut surface.

"LIVER"—Amyloid Degeneration.

PREPARATION :—(a) Stain two or three minutes in Methyl-violet, (b) Wash in $\frac{1}{2}$ percent Hydrochloric acid solution, (c) Wash in water, (d) Mount in Farrant.

ETIOLOGY :—Long continued suppurative diseases, especially of bone, also in chronic tuberculosis and in syphilis.

MICROSCOPIC EXAM :—Begins as a thickening of media of intermediate sized vessels, in middle zone of lobules; when advanced whole lobule becomes affected. By ordinary stain, e.g., Haematoxylin and Eosin these degenerated areas take no nuclear stain, but simply the pink back ground stain of Eosin, while the rest of the tissue stains well. With above special method, the degenerated areas take on reddish-violet stain, while the healthy

tissue takes on a blue or violet stain. From swelling and degeneration of capillary walls, the hepatic cells between these vessels are compressed, and they in turn, become atrophied and degenerated—this is well seen in advanced cases of Amyloid degeneration of the Liver.

MACROSCOPIC EXAM :—Organ enlarged, surface smooth and pale colored. On section, liver is firm, smooth, pale colored with some irregular veining of a pearly-white color. If a solution of Iodine be poured over the cut surface the Amyloid areas take on a mahogany-brown color, while the surrounding tissue is of a yellowish color. In the Liver, generally this Amyloid process begins in the smallest capillaries, i.e., about the middle of the lobule.

"LIVER"—Passive Congestion. (Nutmeg Liver.)

ETIOLOGY :—Valvular disease of heart, pericarditis with effusion, emphysema and all conditions leading to increased pressure in the inferior vena cava.

MICROSCOPICAL EXAM :—Dilated central or intralobular vein with a like condition of its surrounding capillaries. These are filled with blood, and the liver cells in the immediate neighborhood of central vein are atrophied and contain granules of brownish pigment. In places these cells are entirely replaced by a fibrous tissue, especially in advanced cases. Cells of periphery of lobules show more or less fatty infiltration.

MACROSCOPIC EXAM:—Organ enlarged, (in early stages), atrophied in later stage, deep colored, and firm. On section, liver bleeds freely, mottled, like a nutmeg, i.e., deep red centre from dilated vein and capillaries, outer zone of lobule is yellowish from fatty infiltration. In advanced cases, organ is firm and fibroid.

"LIVER"—Portal Cirrhosis (Syn.) Atrophic Cirrhosis.

"GIN DRINKERS LIVER," Hobnail Liver.

ETIOLOGY :—Supposedly to toxines or chemical irritants which first reduce the resisting powers of the hepatic cells, associated with an increased number of bac-

teria brought especially through the portal system, from diseased gastro-intestinal tract.

MICROSCOPICAL EXAM :—Great proliferation of connective tissue about the medium sized interlobular vessels. When early, there is proliferation of small round fibroblastic cells in place of fibrous tissue. This proliferation is multilobular i.e., surrounding several lobules, as distinguished from monolobular i.e., surrounding individual lobules. This tissue does not tend to invade the lobules. Cells in neighborhood of connective tissue are shrunken, atrophied and degenerated from pressure. This contraction also leads to obstruction of portal veins, rarely of biliary ducts. May get fatty infiltration, associated with this condition.

MACROSCOPIC EXAM :—In earlier stages, organ is enlarged, but in advanced cases it is atrophied, hard, granular or irregular and uneven on surface. On section, firm and granular with dull grey or white bands of connective tissue running through organ, these bands enclosing areas of comparatively healthy liver tissue, which is squeezed beyond surface of section by fibrous contraction. This latter tissue may be yellowish or brown from degeneration.

"LIVER"—Hypertrophic Cirrhosis.

ETIOLOGY :—As in atrophic form, more frequent in warm climates.

MICROSCOPIC EXAM :—Proliferation of connective tissue which is monolobular in character. This tissue tends to invade intralobular tissue from periphery towards centre, thus closing off groups or bundles of hepatic cells, which atrophy and degenerate. In these bundles of fibrous tissue may be seen double rows of cells in somewhat tubular form which stain deeply. These are the proliferated bile capillaries which some believe are formed by the double rows of embryonic liver cells. In some cases, there is proliferation of the hepatic cells, but this is rare.

MACROSCOPIC EXAM:—Organ is uniformly enlarg-

ed, surface smooth, moderately granular and firm. On section, yellowish or greenish, either uniform or mottled.

"LIVER"—Biliary Cirrhosis.

ETIOLOGY :—Obstruction to free outflow of bile. From conditions either within or without the common duct narrowing its lumen, e.g., calculi, tumors, etc.

MICROSCOPIC EXAM :—Similar to hypertrophic form. Parenchyma deeply bile stained. First you have spots of insular necrosis in periphery of lobules. This is replaced by fibrous tissue, which later spreads to interlobular tissue. Periangiocholitis (proliferation of fibrous tissue about interlobular bile capillaries) may be characteristic. Where obstruction is sudden or complete, we may get degeneration of the whole organ.

"LIVER"—Pictou Cattle Disease.

ETIOLOGY :—Thought by Dr. Adami, who has made thorough investigations into the etiology and pathology of disease, to be due to minute diplococcoid organisms which are akin to the B. Coli Communis, as in the case of portal cirrhosis. This condition is confined to the environs of Pictou, Nova Scotia.

MICROSCOPICAL EXAM :—Connective tissue proliferation is more extreme than in the ordinary forms, and is of a peri-cellular type, i.e., involving groups of isolated cells throughout the lobules, and not confined to perilobular tissue. The hepatic cells in this variety, show very marked atrophy and degeneration which is best studied by the aid of the high power.

MACROSCOPICAL EXAM :—Shows extreme grade of portal cirrhosis.

"LIVER"—Syphilitic Gumma, with Pericellular Cirrhosis.

ETIOLOGY :—Syphilis.

MICROSCOPICAL EXAM :—As in Pictou cattle disease, the fibrosis is of the pericellular type and is asso-

ciated with thickening of the arterial coats, more especially of the Intima (Endarteritis Obliterans) which leads to occlusion of the vessels. This is a constant character in Syphilis. You may have, in addition, recent gummata, which are somewhat similar to tubercular foci, but which tend early to lead to proliferation of fibrous tissue in periphery of gummatous area, and later, to extreme cicatricial contraction.

MACROSCOPIC EXAM:—As in portal cirrhosis, associated with greater or lesser degree of lobulation of organ. In some cases, you get recent gummata with caseous centres and fibrous capsules. If of long standing, you may get stellate scars, from old healed gummata. Condition is frequently associated with perihepatitis.

"LIVER"—Tubercular Hepatitis.

ETIOLOGY :—Never primary, but associated with general miliary tuberculosis or secondary to tuberculosis of other parts of the body, especially that of peritoneum.

MICROSCOPICAL EXAM :—Note first with low power, the interlobular tissue, or at margin of the lobule, granular masses displacing liver cells, and infiltrating surrounding tissue. In centre of this mass of well stained small round cells, is a crescentic or rounded deeply staining area, about the size of a pin's head, surrounding a clear homogenous centre (which takes on Eosin stain only). With high power, note that peripheral part of this mass is composed almost entirely of small round cells, in meshes of fibrous tissue. In tracing this towards centre, the meshes of fibrous tissue open out and in their meshes are seen, with the small round cells, large avoid cells, having one, two, or more nuclei (Endothelial cells). In centre of this mass is found the Giant cell, which is a large branching cell with peripherally placed nuclei. In centre of this Giant cell, the tissue is homogenous and translucent. As the disease progresses by the formation of new follicles, the centres of which are more and more cut off from nutritive supply, they caseate. In chronic conditions, a fibrous tissue capsule may form about these areas and the caseated material may become organized or impregnated with lime salts and thus become calcified.

MACROSCOPICAL EXAM:—Small greyish nodules either in capsule or in the liver parenchyma. Those soon become caseated and pigmented by bile pigment.

"LIVER"—Abscess.

ETIOLOGY :—In tropical climates as a complication of amoebic dysentery, in pyaemia following upon suppurative conditions in area from which portal system is derived, and from suppurative conditions in gall bladder and bile ducts.

MICROS. EXAM :—May get any degree from simple grouping of leucocytes to complete coagulation necrosis of central portion. In periphery you may have granulation tissue or fibrosis forming a capsule around broken down tissue.

MACROS. EXAM :—In tropics, abscess generally single and situated in right lobe. The contents are a brownish-red pus resembling anchovy sauce. Inner layers of walls composed of greyish ragged necrotic tissue. In metastatic form get multiple minute abscesses throughout organ.

"LIVER"—Pernicious Anaemia.

PREPARATION :—(a) Pass through Potassium Ferrocyanide.
(b) Wash in Hydrochloric acid ½ percent.
(c) Mount in Farrant.

ETIOLOGY :—Part of general anaemia associated with destruction of red blood cells.

MICROS. EXAM :—Deposit of brownish granules of an iron containing pigment in hepatic and endothelial cells of portal zones of lobules, in some the whole lobule is involved—usually only outer two-thirds, the inner third being infiltrated with fat. By this preparation, the iron assumes a Prussian blue color.

MACROS. EXAM :—Organ very pale colored or brownish-yellow and fatty.

"LIVER"—Perihepatitis (Zuckergusslebr of Curschmann).

ETIOLOGY :—Associated with Cirrhosis of liver, chronic peritonitis and especially syphilis.

MICROS. EXAM :—Great thickening of capsule from connective tissue proliferation. This in places extending into parenchyma of organ leading to pressure atrophy and degeneration of cells.

MACROS. EXAM:—Capsule thickened and firm, with, in some cases, threads of tissue attached to capsule. In places thickened capsule dips into interior of organ. Organ at times compressed.

"LIVER"—Hydatid or Echinococcus Cyst.

ETIOLOGY :—Taenia Echinococcus.

MICROSCOPICAL EXAMINATION :—The wall is composed of laminat[illegible] of elastic tissue; adherent to its inner surface are a [illegible]ber of scolices or their hooklets (daughter cysts); the contents are fluid, and may contain these same elements. In the liver these set up an inflammatory reaction producing a fibrosis about the cyst with great proliferation of fibroblastic cells. This fibrosis may lead to pressure atrophy and degeneration of the hepatic cells in its neighbourhood.

MACROSCOPICAL EXAMINATION : — Cysts throughout liver with much thickened capsules, with an inner parenchymatous layer. The contents, clear fluid.

PANCREAS.

"PANCREAS"—Fat-necrosis.

ETIOLOGY :—A form of degeneration common to pacreas and omentum. Due to liberation of fat-splitting ferment of pancreas which acts upon fat cells converting the fats first into fatty acids and then into salts formed by combination of fatty acids with calcium. May be associated with other diseases of this organ.

MICROS. EXAM :—Formation of fatty acids within fat cells with later disintegration of cells and deposit of calcareous salts (Seen as granular translucent masses).

MACROS. EXAM :—Minute, greyish-white, opaque bodies throughout organ and on its surface, with surrounding inflammatory zone. Possibly haemorrhages and disorganization of whole organ.

"PANCREAS"—Acute Haemorrhagic Pancreatitis

ETIOLOGY :—In young persons frequently due to infection through its ducts.

MICROS. EXAM :—Cells show swelling with parenchymatous degeneration. Interlobular tissue infiltrated with small round cells. May get extensive death and necrosis or proliferation of connective tissue.

MACROS. EXAM :—Organ swollen and haemorrhagic, lobules enlarged with compression of interlobular tissue.

"PANCREAS"—Acute Suppurative Pancreatitis.

ETIOLOGY :—Extension of suppurative processes through ducts or from neighboring organs, e.g., perforating peptic or duodenal ulcer, from haemorrhagic form, or from metastases.

MICROS. EXAM :—As in haemorrhagic form, you may get haemorrhagic infiltration in small or large areas. Tissues may all become homogeneous. They stain badly (from degeneration) with here and there areas of small round celled clumping (abscesses). These, later, may break down forming large abscess cavities. Besides the above changes, you also get suppurative inflammation of ducts with thickening of mucosa and filling up of the lumen with muco-pus. The walls of ducts show marked infiltration with small round cells.

MACROS. EXAM :—Organ enlarged, softened with areas of necrosis. Whole organ may be completely macerated from suppuration and pancreatic digestion.

"PANCREAS"—Chronic Indurative or Cirrhotic Pancreatitis.

ETIOLOGY :—Repeated or prolonged acute attacks, Syphilis, senility and obstruction of pancreatic ducts from calculi.

MICROS EXAM :—Tissue between acini shows marked proliferation of fibroblasts and fibrous tissue. Parenchyma atrophied and degenerated. Fat-necrosis and fatty degeneration of cells frequently present. Ducts frequently dilated and cystic with flattening of their epithelial lining. This is especially well marked in the obstructive form. In this form you may have a catarrhal exudate in ducts.

MACROS. EXAM :—Small and firm. On section, hard, gritty of cartilaginous hardiness, cut surface homogeneous, bright bands of connective tissue throughout parenchyma. Possibly cysts throughout organ.

GASTRO-INTESTINAL TRACT.

"STOMACH"—Acute Catarrhal Enteritis.

ETIOLOGY :—Chemical, mechanical, thermal and bacterial irritants.

MICROSCOPICAL EXAMINATION : — Cylindrical cells lining tubules show marked mucous degeneration; the cuboidal cells of the gland fundi are desquamated and degenerated. Between the tubules the blood vessels are engorged and there is marked infiltration with small round cells, these cells may extend into submucor There is also marked hyperplasia of lymph nodes in the mucosa.

MACROSCOPICAL EXAMINATION : — Mucous membrane reddened, possibly haemorrhagic (if very severe) and covered with thick layer of tenacious mucus.

"STOMACH"—Chronic Catarrhal Gastritis.

ETIOLOGY :—Continued or repeated acute attacks,

irregularity and abuse in eating and drinking, and chronic venous congestion as in portal cirrhosis.

MICROSCOPICAL EXAMINATION :—As in the acute form you have mucous degeneration of cylindrical cells with proliferation, desquamation and degeneration of the cuboidal cells of gland fundus. The vessels throughout the interglandular stroma and those of submucosa are congested, their walls are thickened and around them there is a marked proliferation of fibroblastic cells in various stages of development. The glands may become cystic, their lumina being filled up with epithelial cells and mucus; later the glands may become completely atrophied with disappearance of the lumina. As this condition progresses there may be a corresponding increase of the fibrous tissue which may later wholly replace the glandular elements. As cicatricial contraction goes on in this new tissue, portions of the mucosa may be protruded forming polypi (Gastritis Polyposa). Some of these polypi may become cystic from blocking of gland ducts.

The fibrous proliferation may involve the mucous, submucous and muscular coats causing an interstitial gastritis or gastro-phthisis.

MACROSCOPICAL EXAMINATION : — Mucosa somewhat granular, thrown into many folds; possibly there may be polypoid elevations. In many cases color of mucosa is grey but from chronic congestion may have a slaty pigmentation. The whole surface may be thickly coated with tenacious mucus. In the interstitial form all the coats may be thickened, firm, and glistening on section and the organ may be markedly contracted.

"STOMACH"—Peptic or Round Ulcer.

ETIOLOGY :—Frequently in chlorotic young women whereever vitality has been much impaired; from cachexias, localised anaemia (tight lacing), and extensive burns, especially of the abdomen. Here the condition is more frequently found in the duodenum.

MICROSCOPICAL EXAMINATION :—Around the ulcer there is marked proliferation of small round cells; these appear to infiltrate deeply the mucosa and submu-

cosa. The gland tubules in the neighborhood may show some proliferation of their epithelial elements. The walls of blood vessels are thickened. If the process is chronic there is formed granulation and connective tissue.

MACROSCOPICAL EXAMINATION :—Usually single and situated at posterior part of lesser curvature. Edges may be ragged and uneven, or if chronic, even and regular. The ulcer is somewhat funnel-shaped with its base towards mucosa. It may perforate, if healed it may form stellate scar, and if multiple these may produce an hour-glass contraction of the stomach.

"STOMACH"—Amyloid Degeneration.

PREPARATION :—As for Amyloid Liver.

Associated with amyloid degeneration of other organs. Generally takes place in submucosa, rarely in mucosa.

"INTESTINES"—Typhoid.

ETIOLOGY :—Bacillus Typhi Abdominalis.

MICROSCOPICAL EXAMINATION :—In the early stages there is great proliferation of the lymphocytes and the endothelial cells, both of the solitary follicles and the Peyer's patches, causing them to swell and bulge into the lumen of bowel pushing before them the tense mucous membrane. These proliferated small round cells may infiltrate slightly the surrounding mucosa and submucosa. Later the surface of these patches becomes necrotic and sloughs off leaving ulcers. These follow the area of the lymph nodes, extend to the muscular coat which forms the base of ulcer. The edges are thin, undermined, being composed of mucous membrane and portions of submucosa. Around the area of ulceration there is a small amount of small round celled infiltration, but not sufficient to cause thickening. When healing takes place the overhanging edges may become adherent to the muscular base and a simple layer of epithelium covers over the surface without any glandular structures. There is no thickening of peritoneal surface. The ulcer may perforate the wall of the bowel.

MACROSCOPICAL EXAMINATION :—The change usually takes place in lower eighteen inches of ileum, but may extend from the mouth to the anus. In the first stage there is swelling of the solitary glands and Peyer's patches forming distinct reddish-grey bulgings into lumen of bowel; the mucosa over them looks pale. Later these patches may ulcerate, this process following the areas of lymph collections, thus forming oval ulcers, their long axis corresponding to that of bowel. The base is smooth, reddish and glistening and formed by the circular muscular coat. The edges are somewhat ragged and undermined and can be readily floated out in water. The surface may be bile stained. The ulcer may extend beyond the area of the Peyer's patches. The serosa is not altered except where a localised peritonitis is set up by an ulcer which is about to perforate.

The mesenteric glands are enlarged and softened.

"INTESTINES"—Tubercular Ulceration.

ETIOLOGY :—Sometimes primary, but most frequently is secondary to pulmonary tuberculosis from swallowing of infective sputum.

MICROSCOPICAL EXAMINATION : — Generally situated in the solitary glands and Peyer's patches, especially of lower part of ileum. The edges of the ulcer are thickened and densely infiltrated with small round cells, which in many cases are composed of many tubercular follicles, and which show some commencing caseation in their central part. Some show the typical giant cell system as described in the Liver. The floor of ulcer which is composed of similar nodules, is rough and granular. These nodules may extend both laterally and into muscularis. In subserous lymphatics similar caseous tubercular nodules may be made out.

MACROSCOPICAL EXAMINATION :—These tubercular ulcers first appear as yellowish caseous nodules in gland ducts and submucosa, which, later breaking down, may have the following characters:—The base, which is formed by the circular muscular coat, is rough and granular; the edges are heaped up but not undermined; their

long axis extends around the bowel, in the course of the lymphatics. Sometimes two or more ulcers may encircle the bowel with thickened portions of mucosa separating them, thus giving them a scalloped appearance. The nodules in the subserous lymphatics are evidenced by yellowish masses corresponding with the base of the ulcer and these caseous areas radiate from this point around the bowel, giving it a stellate form.

If the tissue between base of ulcer and ser sa is felt between finger and thumb, the nodules can be made out as shotty bodies.

"INTESTINES"—Tropical Dysentery.

ETIOLOGY :—Due to Amoeba Coli. Affects especially the large intestine.

May get petechial haemorrhages with follicular ulcers. The ulcers may tend to become chronic causing thickening of the bowel or there may be a gangrenous form where the epithelial cells, embedded in muco-fibrinous matrix, show granular degeneration and necrosis. The mucosa and submucosa are infiltrated with small round cells.

"INTESTINES"—Amyloid Degeneration.

ETIOLOGY :—Associated with amyloid degeneration of Spleen, Liver and Kidneys. It is often the cause of the watery diarrhoea.

MICROSCOPICAL EXAMINATION :—The amyloid material is laid down in the capillaries of the villi; some of the larger vessels are also affected, and the connective tissue fibrils of muscularis leading to atrophy and degeneration of muscle cells.

MACROSCOPICAL EXAMINATION : — At lower part of jejunum and upper part of ileum the mucosa becomes pale, smooth and translucent. If solution of iodine is poured on the surface mahogany-brown areas, corresponding to villi, are seen, the rest of the tissue being yellow in color.

"APPENDIX VERMIFORMIS"—Appendicitis.

ETIOLOGY :—Secondary to a catarrhal inflammation of the intestines; or to injury from foreign body, or faecal concretion within its lumen, together with bacterial infection, e. g., from the Bacillus Coli Communis.

We may have an acute catarrhal type (Acute Catarrhal Appendicitis), corresponding to that found in stomach, with thickening of mucosa, small round cells infiltrating mucosa, submucosa, and, at times, all the coats; hyperplasia of solitary follicles and congestion of vessels, especially those of serosa. There may be punctate haemorrhages if condition is very severe. This process may pass on to an ulcerative type (Ulcerative Appendicitis), or to a gangrenous form where all the coats are densely infiltrated with pus cells. The arterial supply is cut off from thrombosis of appendicular vessels and the whole tissue becomes necrotic (gangrenous appendicitis).

Again there may be a marked proliferation of connective tissue throughout all the coats with atrophy and degeneration of mucosa. If the muscularis is involved the fibroid thickening is most marked (Chronic Interstitial Appendicitis or Sclerosing appendicitis).

RESPIRATORY SYSTEM.

LUNGS.

Acute Pleuritis (Pleurisy).

ETIOLOGY:—As in Pneumonia, and accompanying it.

MICROS. EXAM :—During the first stage, i.e., (Congestion), the vessels in the pleura, especially in the superficial layer, are congested with slight exudation of leucocytes around them. On the surface, a very thin layer of fibrin with a few small round cells is seen. The endothelial cells of the pleura are swollen and cloudy. In the second stage, i.e., effusion, congestion of the pleural vessels and of the subpleural vessels is marked. Around these, there is a marked round celled infiltration. The

endothelial cells on the surface have become swollen, proliferated and desquamated, and on this denuded area is situated a thick layer of fibrin, in the meshes of which are vast numbers of small round cells. A certain amount of pneumonia may be present, or it may be associated with a pneumonia.

In the third stage (organization) small loops of vessels may be seen penetrating the exudate on the surface. Around these are aggregated many fibroblastic cells, which later become fibrous tissue cells. The fluid part is absorbed and the whole exudate thus becomes organized connective tissue.

MACROS. EXAM :—In first stage there is general redness and congestion with oedema and opacity of pleural surface. With second stage you have well formed exudation of inflammatory lymph as a whitish opaque membrane which can be readily scraped off the roughened pleural surface. Serum may also be present in the pleural cavity. In the stage of organization there is a dense white glistening fibrous membrane adherent to the surface of pleura, and both parietal and viceral layers may be bound together either as one membrane by this, or loosely by thin bands of fibrous tissue.

Tubercular Pleurisy.

The process here is likely to be a chronic one, the principal features being the slow growth, the great thickening of the membrane with caseation and giant cell formation as in tuberculosis of other regions.

"LUNG"—Acute Lobar, Fibrinous or Croupous Pneumonia

ETIOLOGY :—Exposure to cold with Pneumococcus infection.

The folowing four stages may be recognized :—

1st. stage.—Congestion.
2nd. stage.—Red Hepatization.
3rd. stage.—Grey Hepatization.
4th. stage.—Resolution.

"LUNG"—Congestion.

MICROS. EXAM :—Vessels throughout lung are

markedly congested and filled with red blood cells. These, in the alveolar walls forming bulgings into alveoli which can be well seen under the microscope. Around these vessels there is slight small round celled infiltration. Some red blood cells may escape into air cells. Epithelial cells of air sacs swollen, cloudy, proliferating and, in places, desquamating into alveolar spaces.

MACROS. EXAM :—Organ markedly congested, especially at base and posterior borders, of a deep red or purplish color, pleural vessels congested, lung substance firmer but more friable. On section, somewhat oedematous of deep red color (bright red on exposure to air), bloody frothy fluid exudes from cut surface. Section floats in water.

"LUNG"—Red Hepatization.

MICROS. EXAM :—In this specimen note that air cavities are filled up with the following exudate :—The bulk is formed of coagulated fibrin in the meshes of which are large numbers of red blood cells with few leucocytes, and still fewer large epithelial cells. This exudate completely fills and distends the air spaces. Blood vessels in the alveolar walls are less marked from compression of the exudate. Vessels in the deep layer of the pleura and in the interlobular septa are congested, especially those of the lower lobe. With the above changes you may also get some associated pleurisy. (See Pleurisy).

MACROS. EXAM :—The whole lung, or one or more of its lobes, especially the lower lobe, is completely solidified, firm and noncrepitant. On section bright red, firm, granular and noncrepitant, sinks in water. Pleura may show a layer of inflammatory lymph or mere congestion of its superficial vessels. On squeezing only a small amount of blood exudes from cut surface. The rest of lung is congested.

"LUNG"—Grey Hepatization.

MICROS. EXAM :—The air vessels at this stage contain an exudate which is rich in small round cells and only occupies the central portion of the spaces leaving the peripheral portion comparatively clear. The vessels are better marked than in the last stage. The fibrin here has broken down, the red blood cells have, in a great measure, become disintegrated, but there has been an abundant

pouring out of leucocytes, many of which are undergoing degeneration. The whole exudate is granular. In the alveolar walls the vessels are somewhat compressed, and around them is an increased number of fibroblastic cells in all stages of development.

MACROS. EXAM :—The affected portions are as in red hepatization, solid and nonerepitant. On section the tissue is reddish or greyish-yellow, granular and very friable. Section sinks in water. Some muco-purulent material can be scraped off the cut surface. Although the different stages of pneumonia are described here, yet it is very rarely that one can see in any one specimen the typical stages of the disease. More frequently one gets combinations of these various stages, or that of red hepatization passing into that of grey hepatization and so on. The pleurisy progresses with the pneumonia, as a rule.

"LUNG"—Stage of Resolution.

MICROS EXAM :—The exudate now becomes more fatty and degenerated with shrinking from the walls of the alveoli, thus occupying only a small portion of the centre of the air sacs. The vessels have now become prominent and function is completely restored.

MACROS. EXAM :—Tissue is pale, soft and flabby. Mucopus can be squeezed from the cut surface.

Bronchi Acute Catarrhal Bronchitis.

ETIOLOGY :—Measles, whooping-cough, influenza, exposure to cold.

MICROS. EXAM :—Mucous membrane shows marked small round celled infiltration. The epithelial lining cells are, in many cases, undergoing mucoid degeneration, and are desquamating. The vasa vasorum, especially those of subendothelial layer are markedly congested and show surrounding small celled infiltration. This latter infiltration may penetrate all the coats.

MACROS. EXAM :—Thick, tenacious mucous adherent to deeply injected membrane, which, if very acute, may be somewhat odematous, and possibly haemorrhagic.

"BRONCHI"—Chronic Catarrhal Bronchitis.

See Chronic Catarrhal Gastritis, which represents the chronic inflammation of a mucous membrane.

ETIOLOGY :—Repeated or prolonged acute form, emphysema and lung diseases, chronic venous congestion as in cardiac disease.

"BRONCHI"—Fibrinous Bronchitis.

ETIOLOGY —Generally associated with Diptheria of Larynx and Trachea, from inhalation of powerful irritants and in pneumonia.

MICROS. EXAM :—We get fibrinous plugs in bronchi, which may be in the form of fine spirals (Curschmann's spirals).

"LUNG"—Emphysema.

ETIOLOGY :—Chronic Bronchitis, Asthma, old age and general Arterio Sclerosis, and as a compensatory change.

MICROS. EXAM :—The air sacs, from pressure by contained air, become dilated evenly, therefore, they first become enlarged and circular. This leads to pressure of the alveolar walls with their contained vessels causing their atrophy with diminished nutrition to the part. The alveolar walls, therefore, become thin and fibroid, and, in places, ulcerate through leading to intercommunication of neighboring air vesicles. This is the vesicular type. If the air breaks through into the interstitial tissue, you then get the interstitial form. The compensatory form is due to partial emphysema to compensate for some areas of collapse or consolidation of lung. You frequently have an associated arterio-Sclerosis.

MACROS. EXAM :—The lungs are more voluminous, edges and apices are the areas most involved, and these are light in color and in weight, their lobules are well marked out and enlarged. These areas feel like down pillows. On section, tissue is gritty, dry and harsh. Frequently, mucopus exudes on squeezing, from the bronchi. The senile form shows marked decrease in the size of the lung. The epithelial cells lining air vesicles are compressed and flattened.

"LUNG"—Collapse.

ETIOLOGY :—In extreme exhaustion prior to death, from effusions into pleural cavity, plugging of bronchi.

wounds in chest wall, and in weakly new born infants (atelectasis).

MICROS. EXAM :—The lung tissue shows no distention of air vesicles, but is composed rather of a solid mass of interstitial tissue or alveolar walls with the interlobular septa. In the atelectatic form, the epithelial cells lining air vesicles are not compressed and flattened as in those who have breathed, but they are more cuboidal in shape, and occupy a greater portion of the lung tissue.

MACROS. EXAM :—The lung is deep colored, sometimes purplish, firm, fleshy, non-crepitant and sinks in water.

"LUNG"—Lobular, Catarrhal or Broncho Pneumonia.

ETIOLOGY :—Usually follows upon a bronchitis, more especially the capillary form. It is peculiarly frequent in senile emphysema and in measles.

MICROS. EXAM :—Only portions of the lung substance are solidified, and these areas correspond with the distribution of certain bronchi. There is an associated acute bronchitis. The exudate, instead of being fibrinous is catarrhal in type, i.e., it is composed of mucus with large numbers of large epithelial cells (supposedly from lining epithelium) with leucocytes and few red blood cells. The neighboring alveoli show some degree of compensatory emphysema.

MACROS. EXAM :—Only certain areas or lobules of the lungs are involved—these stand out beyond the surface of the lung as deep purplish areas, which are noncrepitant, firm and sink in water. This form is not usually accompanied by pleurisy.

"LUNG"—Chronic Venous Congestion. Cardiac Lung or Brown Induration.

ETIOLOGY :—Valvular heart disease, especially of mitral valve, and in aortic disease.

MICROS. EXAM :—The vessels in the alveolar walls, the interlobular septa and deeper layer of the pleura are markedly congested. In the alveolar walls, they appear to be beaded or varicose. The alveolar walls are thickened, and in them there is a considerable amount of blackish or golden pigment (in the endothelial cells of lymph

channels and in connective tissue cells). Within the air vesicles there are numerous large oval uni-and multi-nucleated cells in many of which there is a deposit of golden-brown pigment (Cardiac cells supposedly derived from the cells lining the air vesicles). With these cells there are many red blood cells. The vessels in the alveolar walls are thickened from fibrous tissue proliferation. Epithelial cells lining alveoli are rounded and swollen, and frequently proliferating and desquamating. The bronchial mucosa is congested with mucopurulent exudation into lumina. The whole organ may become fibroid. Pleura is frequently thickened.

MACROSCOPICAL EXAMINATION :—Lung enlarged and heavy. Pleura has reddish-purple hue. Whole lung is somewhat hard, firm, and of a deep brownish color, especially along the lines of interlobular septa. A large amount of frothy serum exudes from cut surface. Lung is crepitant and section floats in water. Bronchial mucosa congested, oedematous, folded and corrugated looking.

"LUNG"—Infarct.

(See Infarct of Kidney).

One may, in this organ, get "fat emboli" from fracture of bones, especially of cranial bones. The infarct is usually a "red infarct" in the lung, though a "white infarct" may occur.

"LUNG"—Fibroid Pneumonia, or Chronic Interstitial Pneumonia.

ETIOLOGY :—Usually follows upon acute form; thickening of pleura and compression of lung, as from chronic pleuritic effusion.

MICROSCOPICAL EXAMINATION :—The connective tissue frame work of the lung and the walls of the alveoli show marked fibrous proliferation with cicatrgicial contraction, obstructing and obliterating the bronchi and air vesicles. If it is due to non-resolution of a fibrinous exudate, then new capillaries are thrown out into the exudate, with later proliferation of fibroblastic cells and organisation of exudate; if condition is secondary to a thickening of the pleura, then broad bands of fibrous tissue invade the lung substance along the lines of the interlobular

septa. In some cases the bronchi are partly occluded by the cicatricial contraction and the distal portions become dilated (bronchiectasis).

MACROSCOPICAL EXAMINATION :—Lung smaller than normal, firm and subcrepitant. Visceral pleura thickened and densely adherent to parietal layer. Lung tissue at border emphysematos. On section tissue is firm and fibrous; pleura thickened and extending down from it into lung tissue are bands of whitish glistening connective tissue. Cavities (bronchiectatic), are found along the course of the bronchi.

"LUNG"—Pneumono-Konioses or Dust Diseases.

VARIETIES :—The following are the most important :—

1. Anthracosis or Coal-Miner's Phthisis.
2. Silicosis, Chalicosis or Stone-Mason's Phthisis.
3. Siderosis, or Needle-Grinder's Phthisis.

1.—ANTHRACOSIS.

ETIOLOGY :—The long-continued inhalation of coal dust into lungs.

MICROSCOPICAL EXAMINATION : — Coal-black pigment is found in the following positions, viz.: in the interalveolar and interlobular septa, in deep layer of pleura, in the perivascular and peribronchial tissue and within the air cells, either lying free in cavity or included in the epithelial or phagocytic cells. When the pigment is inhaled it is brought by the phagocytes into the lymph spaces in the above mentioned areas, and is then deposited along these spaces and in the connective tissue cells. These irritating particles produce an inflammatory reaction with congestion, and proliferation of fibroblastic cells, thus leading, in time, to an indurative condition with thickening of the walls of the blood vessels. When the intima becomes thickened the tissue thus deposited loses its nutritive supply causing a necrosis of the centres of these fibrous nodules. Microscopically these areas then are recognized by their caseous centres wi h surrounding pigmentation and proliferation of connec ve tissue. The bronchi show a condition of bronchitis.

MACROSCOPICAL EXAMINATION : — The whole lung may be black in color, enlarged, firm and fibroid. The deeper layer of pleura is much thickened and studded with fibroid or caseous nodules with deep black pigmentation. The same changes take place along the lines of the interlobular septa and the perivascular and peribronchial tissues, especially the former. The peribronchial lymph glands are pigmented, enlarged and fibroid, frequently caseous.

2. SILICOSIS. 3. SIDEROSIS.

In these conditions the same changes take place, but the pigment is of a lighter color and the particles being far more brittle and irritating, the processes of induration and caseation are much more marked than in anthracosis.

" LUNG "—Disseminated Miliary Tuberculosis.

ETIOLOGY :—In general miliary tuberculosis or extending from local foci in lungs and pleurae.

MICROSCOPICAL EXAMINATION :—The tubercular foci are found, as described, in the liver, situated in the interlobular septa and the alveolar walls. From the thickening of the alveolar walls the air sacs are compressed and collapsed.

MACROSCOPICAL EXAMINATION : — Scattered over the surface of the lungs are small translucent greyish masses about the size of small shot, the intervening lung tissue is red and congested; these may be situated throughout the lung. If they are of haematoginous origin then they are generally situated beneath the pleura, if of bronchogenic origin they generally are arranged about certain bronchi. When they are secondary to tuberculous pleurisy they are situated in deeper layers of pleura and along the interlobular septa.

" LUNG "—Chronic Tuberculosis with Cavitation.

If a portion of lung tissue with the included cavity be examined the following appearance may be seen :—

MICROSCOPICAL EXAMINATION : — The central portion of cavity may consist of cheesy homogeneous non-staining caseous material; the central portion of this tissue may have been expectorated, leaving a central space

with area of surrounding caseation. In the periphery of this caseation there are new giant cells (already described in the Liver) with a marked proliferation of small round and endothelial cells. At the outermost part of this layer there is a well marked proliferation of connective tissue, forming a capsule. The central necrotic tissue may be infiltrated with lime salts forming a deeply staining granular mass. New tubercles may be formed throughout the lung, especially in the peribronchial, perivascular and interlobular lymphatics, and in deep layers of pleura, which latter membrane shows marked tubercular involvement. The intervening lung tissue frequently becomes consolidated, producing condition known as acute pneumonic phthisis.

MACROSCOPICAL EXAMINATION : — Lung is densely adherent to chest wall. Pleura thickened and presents on its surface bluish, yellowish or greyish caseous masses. Lung tissue is firm and nodular, while on section, the pleura is found much thicker with areas of caseation. The same condition is found along the interlobular septa. Throughout the organ there are cavities ranging in size from that of a walnut to that of an orange. They contain yellowish cheesy pus, their walls are thickened with ragged masses adherent to the inner surface which is deeply pigmente d. Across the cavities are numerous bands, corresponding to the vessels and interlobular septa, giving the cavity a honeycombed appearance. Around these cavities there is a well marked fibrous capsule. The contents of a cavity may be organized connective tissue or they may be infiltrated with lime salts forming calcareous nodules. The peribronchial lymph glands are enlarged, pigmented and caseous.

PYAEMIC ABSCESSES may be met with in the lung in a general pyaemia or from an unresolved pneumonia.

GANGRENE may also occur as a complication of pneumonia.

CIRCULATORY SYSTEM.
HEART.

"HEART"—Cloudy Swelling.

See cloudy Liver.

"HEART"—Fatty Infiltration.

PREPARATION :—As for fatty Liver.
ETIOLOGY:—Causes producing general obesity.
MICROS. EXAM :—Adipose tissue in subpericardium increased in amount. This tissue invades the myocardium as fatty bands between muscle bundles. The fibres, if affected at all, show pressure atrophy.
MACROS. EXAM :—Subpericardial fat is much increased in amount. On section, yellowish bands of fat can be seen to dip down from pericardial surface between the darker bands of the heart muscle.

"HEART"—Fatty Degeneration.

PREPARATION :—As for Fatty Liver.
ETIOLOGY:—Following cloudy swelling, and cases of malnutrition, e.g. in severe anaemia or arterio-sclerosis of coronaries, bacterial and chemical toxines, e.g. Septicaemia, Phosphorous poisoning, etc.
MICROS. EXAM :—Cells show same changes as in fatty degeneration of Liver.
MACROS. EXAM :—Muscle is pale colored (in patches), soft, friable and flabby. Pallor especially marked towards endocardial surface, in musculi papillares and columnae carnae. Muscle often resembles faded leaves.

"HEART"—Acute Endocarditis.

ETIOLOGY :—Acute articular rheumatism.
MICROS. EXAM :—At the attachment of the vegetation to valve are great numbers of small round cells, which also infiltrate the subjacent endocardium. On sur-

face of vegetation there is a layer of fibrin in meshes of which many blood corpuscles are entangled. Endothelial cells at point of contact are desquamated and degenerated.

MACROS. EXAM :—On free edges of valve —especially those of left side of heart—and at points of contact, are situated soft, friable, warty-looking masses, which, when torn, leave an ulcerated base on the valve surface.

"HEART"—Chronic Endocarditis.

ETIOLOGY :—Follows acute form, secondary at times to general arterio-sclerosis and atheroma.

MICROS. EXAM :—At base of vegetation have, in place of granulation tissue, well marked proliferation of connective tissue with few fibroblastic cells. This causes thickening of endocardium. On surface of vegetation fibrin and blood cells are also seen as in the acute form. If calcareous degeneration has taken place then you get in deeper layers of this fibrous area a granular deeply staining highly refractive mass which is the calcareous salts deposited in the fatty acids.

MACROS. EXAM :—Vegetations in this form are hard, firm and fibroid, valve segments puckered, at times bound together, and incompetent. Chordae tendinae shortened and thickened. You may get yellowish fatty areas in thickened valve, or in later condition, calcareous plates.

"HEART"—Acute Ulcerative Endocarditis.

ETIOLOGY :—Infective type of acute endocarditis. Differs from the simple form in the greater amount of necrosis of endothelium and in the lesser reparative processes.

MACROS. EXAM :—Necrosis of the endothelium takes place leading to formation of ulcers on which secondary deposits of fibrin may form.

"HEART".—Brown Atrophy.

ETIOLOGY :—Long continued exhausting diseases, e.g., phthisis and Addison's Disease; as a senile change.

MICROSCOPIC EXAMINATION : — Muscle fibres are thin; the bundles of muscular tissue are widely separated. Transverse striation is marked. There is a deposit of pigment at both poles of cell nuclei of a golden or brownish color. This pigment is best seen in unstained specimens.

MACROSCOPIC EXAMINATION : — Heart small, walls thin, muscular tissue of a dark or brownish color, of firm consistence or soft and fatty; orifices somewhat narrowed (more especially the mitral orifice in senile atrophy).

"HEART"—Hypertrophy.

ETIOLOGY :—Valvular disease, emphysema, excessive eating and drinking (especially beer drinking), overwork and strain, diseases of arteries and kidneys.

MICROSCOPIC EXAMINATION :—Individual fibres much thickened, their nuclei (which also appear large) occupying but a small area of cell protoplasm.

MACROSCOPIC EXAMINATION : — Heart walls and musculae papillares are much thickened and firm.

"HEART"—Acute Myocarditis.

Two forms, viz.:—
(a). Circumscribed.
(b). Diffuse.

Acute Circumscribed Myocarditis or Abscess of Heart :

ETIOLOGY :—As an extension from endocardial or pericardial diseases; or from infection through coronary arteries, as in septicaemia, pyaemia and ulcerative endocarditis.

MICROSCOPICAL EXAMINATION : — The heart muscle may be studded with large numbers of minute abscesses, or there may be but a single pus collection.

MACROSCOPICAL EXAMINATION : — There is either a single abscess of heart muscle or multiple minute haemorrhagic or necrotic areas, varying in size from that of a pin's head to that of a cherry. These abscesses may open into endocardium or pericardial sac.

Acute Diffuse Myocarditis.

ETIOLOGY :—Occurs in infectious fevers and general infection.

MICROSCOPIC EXAMINATION :—The connective tissue between muscular fibres is densely infiltrated with small round cells. Later these may lead to proliferation of connective tissue. The blood vessels are engorged; the muscle fibres are granular and opaque with indistinct striation, frequently vacuolated and segmented.

MACROSCOPICAL EXAMINATION :—Muscle lighter in color, soft and friable, frequently haemorrhagic. Heart cavities dilated.

"HEART"—Chronic Myocarditis.

May be circumscribed or diffuse.

ETIOLOGY :—Following acute; secondary to arteriosclerosis and disease of coronaries; secondary to disease of heart valves and pericardium.

MICROSCOPICAL EXAMINATION :—Areas of connective tissue throughout the heart between muscle fibres; pressing these apart and causing pressure atrophy with granular and fatty degeneration of muscle fibres. If process is early there is simply a proliferation of fibroblastic cells between muscle fibres.

MACROSCOPICAL EXAMINATION :—Throughout heart muscle there are white or greyish glistening bands of connective tissue running in the lines of the muscle fibres. This is especially well seen in the papillary muscles. There is possibly some fatty degeneration of heart muscle.

"HEART"—Pericarditis.

See Pleurisy.

BLOOD VESSELS.

"ARTERIES"—Acute Suppurative Arteritis.

ETIOLOGY :—From suppuration surrounding vessels, or from infected emboli.

MICROS. EXAM :—When from without the vessels, there is marked small round celled infiltration with collections of these (pus cells) in adventitia and media. The process may extend to intima.

When from within, there is desquamation and degeneration of endothelium with necrosis of intima, also infiltration of media and adventitia with small round cells.

"ARTERIES"—Acute Productive Arteritis.

ETIOLOGY :—From surrounding connective tissue proliferation and secondary to thrombosis within (thrombo-arteritis).

MICROS. EXAM :—The intima and adventitia are densely infiltrated with small round cells, which also infiltrate the thrombus. Later you get fibrolastic cells with the penetration of new vessels from the vasa vasorum. Thus thrombus becomes organized.

Macroscopically, the thrombus is seen to adhere firmly to vessel wall.

"PERIARTERITIS NODOSA" seen as nodular thickenings of adventitia.

"ARTERIES"—Arteriosclerosis, (syn.) Endarteritis-Deformans or Atheroma.

ETIOLCGY :—In aged, and from continued high pressure pulse, as in chronic Bright's disease.

MICROS. EXAM :—Change takes place within the internal elastic lamina, but involves the wall unequally, giving to it a signet ring appearance. The muscular wall loses its elasticity with consequent localized dilatations, —to counteract this, there is hyperplasia of connective tissue in the intima, which tends to restore the normal caliber. The deeper layers of this hyperplastic tissue may undergo, 1st., coagulation necrosis, then fatty degeneration, and lastly, it may become impregnated with lime salts. This process may be diffuse or nodular. If atheromatous plaque ulcerates through into the lumen, you get an atheromatous ulcer formed.

MACROS. EXAM :—Scattered irregularly over the inner surfaces of Aorta and larger vessels, and especially at the orifices of their branches are numerous, flat, rounded or oval projections of somewhat yellowish color. These may undergo calcareous degeneration. In diffuse form the change takes place especially about the vasa vasorum. In smaller vessels, there are the following differences:— 1st. A simple increase of endothelial cells, or 2nd, obliterating condition from fibrous proliferation beneath the endothelium (endarteritis obliterans) seen in Syphilis and Bright's Disease. You may have in old age, a general calcerous deposit around the whole vessel. These may break down, forming atheromatous abscesses, or if they break through into the lumen of the vessels, to atheromatous ulcers.

VEINS.

These show the same changes as are seen in the arteries.

URO-GENITAL SYSTEM.

KIDNEY.

"KIDNEY"—Cloudy swelling.

See cloudy swelling of Liver. In this case the swelling of epithelial cells is more distinct leading to narrowing of lumen which is represented by small triangular slit.

"KIDNEY"—Fatty Degeneration.

ETIOLOGY :—See Fatty degeneration of liver.

PREPARATION :—See Fatty degeneration of liver.

MICROS. EXAM:—Cells of convoluted tubules swollen and filled up with minute droplets of fat. Similar condition of epithelial cells lining Bowman's capsule. Not so marked in straight or collecting tubules.

MACROS. EXAM :—If advanced, organ is smaller and paler than normal, capsule strips with ease, surface smooth and mottled yellowish with prominent venae stellatae. On section cortex shrunken, mottled, yellowish, interlobular arteries and glomerular tufts prominent. Tissue edges may be ragged and uneven, or if chronic, even flabby and greasy. Medulla striated yellow and red (the latter being due to prominent vessels of medulla).

"KIDNEY"—Parenchymatous Nephritis or Acute Bright's Disease.

ETIOLOGY :—Mechanical, thermal, chemical and bacterial irritants.

MICROS. EXAM :—In some convoluted tubules, lining cells are cloudy, while in others they are swollen, proliferating (by cell division) and degenerating, showing marked fatty degeneration. Some of the tubules are blocked with these fatty desquamated cells. This is best seen in columnar cells of cortex and boundary zone. Interlobular, afferent and glomerular vessels engorged with blood. Around Bowman's capsule small round celled infiltration has taken place showing beginning interstitial nephritis. Red blood cells have extravascated into tissues in places, the amount depending upon severity of inflammation. Hyaline, blood and fatty casts present in lumen of tubules. Medullary vessels engorged with blood.

MACROS. EXAM :—Organ enlarged, rounded, flabby, capsule tense, pulling off readily from smooth pale, mottled, oedematous surface. Venae stellate prominent. On section, cortex is pale, mottled and swollen, glomeruli and interlobular vessels prominent, possibly haemorrhagic, or whole cortex may be red and injected. Medulla is deeply injected. In very advanced cases of acute Bright's disease, you get a large, pale or fatty kidney, with whole organ mottled pink and yellow.

"KIDNEY"—Scarlatinal Nephritis.

MICROS. EXAM :—Around interlobular, afferent and glomerular vessels there is a marked small round celled infiltration. Beneath capsule and at terminal

points of interlobular vessels—these form wedge-shaped masses with base to capsule. This is due to hyaline degeneration of intima and small round celled infiltration of media of the vessels. Haemorrhages here frequent. Otherwise, the condition is very much the same as in acute parenchymatous nephritis. In severer forms, you get widespread small round celled infiltration. Condition is one of the types of Glomerulo-nephritis.

MACROS. EXAM :—Similar to advanced cloudy swelling with marked congestion and haemorrhagic infiltration.

"KIDNEY"—Amyloid Degeneration.

PREPARATION AND ETIOLOGY :—See Amyloid Liver.

MICROS. EXAM :—Parts affected in order are:—Glomerular capillaries, afferent arterioles, interlobular arteries, the arteries of the medulla, veins and capillaries of cortex, and, lastly, the membrana propria of urinary tubules. These structures are rendered thick translucent and homogeneous, and stained by methy violet take on reddish-violet tint. All epithelial elements may undergo fatty degeneration. May get interstitial increase of both fibroblasts and fibrous tissue.

MACROS. EXAM :—Organ enlarged, firm and elastic, uniformly greyish or waxy, or these may alternate with yellowish areas where fatty change is well marked. With iodine solution amyloid areas stain mahogany-brown.

"KIDNEY"—Chronic Intersitial Nephritis.

ETIOLOGY :—Follows upon acute disordered metabolism, e.g., gout, chronic toxaemia and lead poisoning, bacterial infection, etc.

MICROS. EXAM :—Capsule thickened from fibrous proliferation. Alternating areas of (A) wedge-shaped patches from capsule to the apices at the bases of the pyramids. In centre of each of these is situated interlobular artery which is generally irregular, thickened, and tortuous. Around this is a great proliferation of fibrous

tissue, with fibroblasts and leucocytes. Glomerular tufts are irregularly placed and appear comparatively numerous from cicatricial contraction. Tufts of glomerular vessels, at times, have a trilobed appearance and are contracted. Bowman's capsule is thickened and fibroid. Walls of capillaries in tuft undergo hyaline degeneration, leading to clear, transparent condition of tufts. Tubules in this dense tissue are atrophied, the lumina small, lining epithelium flattened, or cubical, and oftentimes degenerated and desquamated; and (B) Areas which are somewhat oval in shape, between indurated masses—in these there is open work with little fibrous deposit. Tubules frequently distended with urinary contents, epithelial lining is flattened. Medulla not much altered except in extreme grades when it may also be indurated and contracted. Tubules may contain hyline, epithelial, and blood casts.

MACROS. EXAM :—Organ contracted, small, firm, capsule thickened, and intimately adherent to surface, part of which it tears away on peeling off. Surface granular (either coarsely or finely). Color of granular protuberances depends on the amount of blood in the cortex, and degree of fatty degeneration of cells. Usually bright greyish red, sometimes mottled, or yellowish—depressions [illegible] Frequently, cysts of all sizes are seen on [illegible] On section cortex narrowed, tough, granular [illegible] ce, and may contain multiple cysts. Medulla slightly redder than usual and contracted in extreme cases. Pelvis shows large increase of fat around the calices.

"KIDNEY"—Arterio Sclerotic.

The only difference between Cirrhotic and Arterio Sclerotic kidney is that in the latter, the process is practically limited to the walls of the arteries and glomerular vessels, and is slow in its progress, while in the former, i.e., in the Cirrhotic form, fibrous proliferation takes place in the interstitial connective tissue outside the vessel-walls, leading to more advanced induration and contraction of stroma, with more extensive degeneration of secreting epithelium of the tubules.

"KIDNEY"—Embolic Suppurative Nephritis.

ETIOLOGY :—Emboli derived from suppurating foci in the other parts of the body e.g., malignant endocarditis.

MICROS. EXAM :—In neighborhood of the glomeruli bacteria lodge in small capillaries, acting as minute emboli, and leading to infiltration of tissue with leucocytes and red-blood cells and to degeneration of kidney parenchyma. These early abscesses are seen as masses of leucocytes, many being in process of degeneration.

MACROS. EXAM :—Minute yellowish or greyish points throughout periphery of organ, surrounded in places by haemorrhagic infiltration. These later may break down and coalesce forming larger abscess cavities.

"KIDNEY"—Suppurative Pyelonephritis.

ETIOLOGY :—Here secondary to suppuration in urinary passages.

MICROS. EXAM :—Uriniferous tubules from calices infiltrated with pus cells, their cellular elements degenerated and desquamated. Many of these coalesce forming larger pus collections. Superficial portion of medulla and cortex secondarily involved.

MACROS EXAM :—Small linear areas of light or yellowish color in pyramids running from calices to boundary zone. These may form large abscess cavities.

"KIDNEY"—Infarct.

ETIOLOGY :—Emboli in blood stream e.g., from vegetative endocarditis.

MICROS. EXAM :—First you get anaemia in area supplied by plugged vessel, then enormous dilatation of vascular channels and sinuses—this is followed by coagulation necrosis and fatty degeneration of area. If collateral circulation is good you may get haemorrhagic infiltration into whole area from venous back pressure. In the periphery the vessels (supplying the capsule) become engorged and the tissue is densely infiltrated with leuco-

cytes or fibroblasts. This tissue may organize with organization of the whole infarcted area, or it may break down, or it may become impregnated with lime salts.

MACROS. EXAM :—If early, the surface of infarct becomes of a deep purple or brick-red color. On section wedge-shaped with base to capsule, surrounding parenchyma is congested. Centre may be any color, from pale to yellow, according to stage, or may, if advanced and organized, be depressed below the surface, and on section it may be firm, whitish and fibroid. These infarcts are apt to be multiple.

"KIDNEY"—Infective Embolic Infarct.

Same changes as in Infarct take place. To these are added the suppuration from infective material. This suppuration takes place from apex of wedge (where emboli are lodged) and extends towards base.

"KIDNEY"—Leukaemia.

MICROS EXAM :—Along the line of intertubular capillaries of glomerular tuft you get enormous increase of leucocytes. May get these throughout vascular and lymphatic channels of whole organ as in the liver. Tubular epithelium comparatively healthy.

MACROS. EXAM :—Cortex pale and enlarged with haemorhages. Pyramids pink with haemorrhages at their bases.

"KIDNEY"—Syphilitic Gummata (rare.)

Generally situated near surface of cortex. Same as in liver.

"KIDNEY"—Miliary Tuberculosis.

ETIOLOGY :—Part of general or disseminated miliary tuberculosis.

MICROS. EXAM :—Miliary tubercles as in Liver are seen around interlobular arteries with reactive interstitial

inflammation in their neighborhood. Typical giant cells are rarely seen in the kidney. Caseation may go on till small cavities are formed.

MACROS. EXAM :—Greyish subcapsular nodules the size of a pin's head; also throughout periphery of cortex.

"KIDNEY"—Tubercular Pyelo-nephritis.

ETIOLOGY :—From extension of tuberculosis of genito-urinary passage.

MICROS. EXAM :—Tuberculous caseation extends from calices and spreads upwards towards cortex as in abscess.

MACROS. EXAM :—Caseation may involve whole organ producing nodular masses on surface. Walls of cavities are ragged and lined by soft, yellowish, caseous material. Contents a yellowish, cheesy material or yellowish fluid. The whole of the calices may be sloughed off—same process seen in the pelvis of the kidney.

LYMPHATIC SYSTEM.

SPLEEN.

"SPLEEN"—Active Hyperaemia.

ETIOLOGY :—In malarial and typhoid fevers, in infectious diseases, and wherever high temperature is continued.

MICROS. EXAM :—Increased amount of blood in sinuses—increase of lymphocytes, especially in Malpighian corpuscles. Endothelial cells lining lymph channels are in places proliferated—these being often swollen and cloudy. If condition leads to acute inflammation, the endothelial plates in the adenoid network are increased in

number, also the leucocytes accumulate in sinuses, and along the trabeculae. This may go on to abscess formation affecting Malpighian corpuscles.

MACROS. EXAM :—Organ enlarged, capsule tense. On section, dark red, soft, diffluent and vascular.

The dark red turns to bright red on exposure to the air. Malpighian corpuscles may stand out as greyish spots. Later stages organ small, capsule wrinkled.

"SPLEEN"—Fibroid.

ETIOLOGY :Repeated or prolonged attacks of febrile diseases, e.g., Malaria, and in rickets and congenital and acquired syphilis.

MICROS. EXAM :—Capsule, trabeculae, walls of sinuses, and adventitia of vessels thickened from fibrous tissue proliferation. Adenoid sheaths of arteries, and malpighian bodies more fibrous with diminution of lymphoid cells. Endothelial cells lining pulp sinuses often contain brownish blood pigment, walls also pigmented.

MACROS. EXAM :—Capsule thickened—organ firm, possibly contracted, thickened fibrous bands running through pulp from capsule. Pulp brittle, of a dirty greyish-red color with possibly brownish pigment.

"SPLEEN"—Chronic Venous Congestion.

ETIOLOGY :—Obstruction to free flow of blood through inferior vena cava, e.g., Emphysema, and valvular disease of the heart, general portal obstruction, e.g., Cirrhosis of Liver, or obstruction of splenic veins.

MICROS. EXAM :—Large venous sinuses engorged with blood, general deposit of fibrous tissue throughout splenic pulp. Adenoid sheaths fibroid, vessel walls thickened, fibrous trabeculae thickened, muscular tissue hypertrophied in capsule and trabeculae. Capsule thickened from localized proliferation of fibrous tissue with shreads of granulation tissue as villous projections from it. Cells lining sinuses often contain altered blood pigment (as in nutmeg liver).

MACROS. EXAM :—Organ enlarged, heavy, and

fleshy—capsule thickened with localized cartilaginoid patches—on section fleshy red or purple. No marked thickening of trabeculae to naked eye.

"SPLEEN"—Infarct.

See Infarct of Kidney.

"SPLEEN"—Waxy—Sago.

PREPARATION AND ETIOLOGY: See Amyloid Liver.

MICROS. EXAM:—Amyloid change is confined almost entirely to the Malpighian corpuscles. The central arteriole and a small amount of adenoid tissue around it are comparatively healthy. The splenic pulp is unaffected. The small arterioles running from Malpighian corpuscles show slight Amyloid degeneration. Throughout splenic pulp are amyloid deposits in small arterioles. With high power note that the capillaries of Malpighian corpuscles are seen as thin homogeneous reddish-violet lines between which are seen the lymphoid cells.

MACROS. EXAM:—Organ is slightly enlarged, firm and elastic. On section, Malpighian bodies stand out on somewhat reddened back-ground as gelatinous masses which resemble boiled sago—with Iodine solution these masses take on mahogany-brown tint.

"SPLEEN"—Diffuse Waxy.

PREPARATION AND ETIOLOGY:—See Amyloid Liver.

MICROS. EXAM:—Fibres of trabeculae about the sinuses, yellow elastic tissue of venous sinuses, the central arteriole of Malpighian body with the tissue in its immediate neighborhood are the parts involved. The rest of Malpighian body is generally fibroid. Endothelial cells and cells between sinuses show marked fatty degeneration, and some possibly amyloid change, though this is doubted by many.

MACROS. EXAM:—Organ much enlarged, edges

rounded, substance firm and elastic. On section, surface transparent and gelatinous. Get mahogany-brown reaction on addition of Iodine solution.

"SPLEEN"—Leukaemia.

ETIOLOGY :—Constant leucocytosis in Leukaemia.

MICROS. EXAM —Splenic pulp consists of one mass of leucocytes. The malpighian bodies may be hypertrophied and fibroid. Sinuses are enormously distended with these leucocytes.

MACROS. EXAM :—Organ evenly enlarged, notches on border prominent, pale colored and firm. Subcapsular haemorrhages, with possibly infarcts. On section, homogeneous, firm, with or without prominent malpighian bodies.

"SPLEEN"—Tuberculosis.

ETIOLOGY :—Seldom or never primary, generally part of a miliary tuberculosis or secondary to this disease in other parts of the body.

MICROS. EXAM :—Appearances are the same as in other organs. You may get either minute miliary tubercles with clumping of small round cells, or more advanced with tuberculous caseation.

MACROS EXAM :—Minute greyish or yellowish translucent bodies beneath capsule or near surface of organ. You may get large caseous nodules, about size of pea or cherry—especially in children.

"SPLEEN"—Syphilis.

You may get gummata, usually multiple, with central degeneration and fibrous striation at periphery.

Diffuse fibrous hyperplasia frequent and almost constant with congenital Syphilis.

"LYMPHATIC GLAND"—Tuberculosis.

ETIOLOGY :—The Bacillus of Tuberculosis reaches the gland through the afferent lymphatics, occasionally through the blood stream.

MICROSCOPIC EXAMINATION :—Small tubercles as in other organs containing giant cells and epithelioid cells with caseation as a more advanced change. These may be surrounded by a zone of small round cells. In some few cases we get simply large increase of epithelioid cells with few small round cells. These latter do not tend to caseate.

MACROSCOPIC EXAMINATION :—Small greyish caseating nodules throughout an enlarged firm gland. The whole gland may be converted into a caseous mass. Capsule frequently thickened and adherent to surrounding structures if condition is advanced.

DUCTLESS GLANDS.

ADRENALS OR SUPRARENAL BODIES.

ADRENALS :—Atrophy is associated with Addison's disease, and is also seen in old age.

ADRENALS :—Haemorrhages are generaly the result of injury.

ADRENALS :—Cloudy swelling is associated with febrile conditions.

ADRENALS :—Fatty infiltration is both physiological and pathological, the latter variety is well seen in marasmic infants. The fat occurs in large globules in the cells of the cortex.

ADRENALS :—Amyloid Degeneration is associated with amyloid degeneration of other organs and takes place in the vessels running vertically through the cortex, possibly in the medullary vessels, but in a less marked degree.

Macroscopically the organ is enlarged, on section translucent. With Iodine solution, the mahogany-brown reaction may be produced.

ADRENALS :—Cysts are rare, examples of these are found in echinococcus cyst, and in haemorrhagic infiltration.

ADRENALS :—Tuberculosis is here found in two forms, viz.—As Miliary Tuberculosis in the cortex, or as a primary chronic tuberculosis of the Medulla. This is the commonest lesion found in Addison's disease.

ADRENALS :—Syphilis is seen in the form of gummata.

ADRENALS :—Actinomycosis is generally secondary to that of the Liver.

THYROID GLAND.

Goitre or Adenomatous Goitre is a simple hyperplasia of gland acini of the normal type. You may get increased secretion of colloid material causing cystic dilatation of acini (cystic or colloid goitre or struma) and flattening of the epithelial cells. Some acini may open into others forming large cysts. We rarely get colloid material in stroma of gland. The capsule and stroma in some cases of goitre is fibroid (Fibroid Goitre). The vascular channels may dilate—hence have a (vascular Goitre.)

GENITAL SYSTEM.

MALE GENITALS.

TESTICLES :—Tuberculosis.

ETIOLOGY :—Either primary in testicle itself, or as most frequently occurs, in the epididymis. The latter may be secondary to tuberculosis of the lower urinary or genital passages; e.g., the bladder, prostate, seminal vesicles, or through the blood channel from tuberculous foci in other parts of the body.

MICROSCOPICAL EXAMINATION :—Caseous masses form in walls of epididymis, which spread, invading all the tissues, through the lymphatic channels. When through the blood current, the tubercles are in the miliary form, as seen in other organs, these rarely extend to testicle itself.

MACROSCOPICAL EXAMINATION :—Tuberculosis in the testicle is a progressive caseating disease forming large caseous masses. There is no tendency to formation of scar tissue as in syphilis. Generally primary in epididymis, but it may extend to tunica vaginalis and testicle.

TESTICLES.—Syphilis.

ETIOLOGY :—In both congenital and acquired forms most frequently occurs as an indurative (fibroid) type, or more rarely as gummata.

MICROSCOPICAL EXAMINATION :—As in other organs the fibrosis is very general, causing marked atrophy of all glandular structures, thickening of vessel walls, particularly of intima (endarteritis obliterans).

MACROSCOPICAL EXAMINATION :—The process usually starts in the testicle itself, extending secondarily to epididymis. The disease is particularly apt to cause stellate scars, particularly in the gummatous form. Thus differing from tuberculosis.

PROSTATE.—Prostatitis.

May get a simple prostatitis associated with a posterior gonorrhoeal urethritis, or a suppurative or phlegmonous prostatitis (abscess). These do not differ from like conditions in other organs.

Concretions may form in the gland tubes as colorless or brownish-black, or even black particles resembling snuff.

Microscopically these form in the gland tubes small oval or rounded deeply staining lamellated masses, called erroneously corpora amylaceae. They are caused by hyaline degeneration of the epithelial cells lining tubes. Later they may become impregnated with lime salts.

Atrophy occurs in old age and in chronic inflammation, and, therefore, shows replacement of glandular elements by fibrous tissue. These bands of fibrous tissue by contraction may cause constriction of gland tubes with cystic dilatation of their distal ends.

Hypertrophy generally occurs in old age and frequently as a complication of a chronic posterior gonorrhoeal urethritis. Proliferated tissue is generally of a fibromyomatous nature.

FEMALE GENITALS.

OVARIES.

Simple inflammation or oophoritis is generally secondary to inflammation of neighbouring parts, e.g., perito-

neum, Fallopian tubes, etc. This may terminate in abscess formation or in general fibrosis. These conditions show same microscopic changes as in other organs. The organ becomes atrophied and fibroid after the menopause.

Tuberculosis, either primary or secondary to adnexa, may form large masses or miliary tubercles. Cysts may form from distension of Graafian follicles with fluid, especially with chronic oophoritis. Colloid or myxoid cysts may form as two different varieties, viz.:—

(a). GLANDULAR CYSTOMATA :—May form from embryonal rests or surface epithelium. Microscopically they form regular gland acini. These have lining of epithelial cells; they may project into cysts whose contents are of a mucoid or colloid character (adenocystoma, or cystadenoma). Macroscopically they form multilocular cysts filled with gelatinous fluid. Some small cysts may project as daughter cysts from walls of larger cysts.

(b). PAPILLARY CYSTOMATA :— Probably take their origin from the paroopheron. May get them a large cysts with papillomatous (see papilloma) projections of lining membrane into them, or they may form papillomatous cystic projections from surface of ovary. Inner lining of cysts and papillomatous projections are composed of stratified ciliated epithelium. Macroscopically the surface variety forms as cauliflower-like projections.

DERMOID CYSTS :—In this form you may get simple cysts containing only epidermal structures, while in others there may be epi, hypo and mesoblastic elements. The contents are thick greyish material (of fatty detritis, degenerated epithelial cells and cholesterin crystals with hair, teeth, etc.)

FALLOPIAN TUBES.

Salpingitis is generally secondary to an inflammation of uterine mucous membrane or to localised peritonitis. May get a simple inflammation (acute catarrhal salpingitis), not differing from other inflammations of the mucous membranes; this may go on to suppuration (suppurative salpingitis).

From a prolonged acute attack or repeated acute inflammations the condition may become chronic (chronic catarrhal salpingitis). This form may be associated with great proliferation of connective tissue.

Tuberculosis (Tubercular salpingitis) may affect this organ as a primary or a secondary disease. e.g., miliary

tuberculosis of peritoneum, or from the lower genital tract by direct extension. The primary form frequently occurs after a gonorrhoeal infection. The mucosa becomes caseous with fibrous thickening of walls of tubes. Microscopically there is complete or partial caseation of mucosa and submucosa with marked small round celled infiltration of all the remaining tissue. Giant cells may or may not be present.

UTERUS.

All the forms of inflammation of this organ (endometritis) are the same as in other mucous membranes.

A form of suppurative inflammation (septic endometritis) is particularly frequent during the puerperium (septic puerperal endometritis).

PLACENTA.

Tuberculosis may occur in this structure as small tubercles in decidua serotina, later invading the placental tissue between the villi.

Syphilis occurs in the diffuse indurative form and as gummata.

BENIGN OR NON-MALIGNANT TUMORS.

FIBROMA.

Occurs on free surface of skin and mucous membranes as papillomata; in the glands or organs as circumscribed nodes. In the former it projects from the surface as finger-like fibroid processes which are covered with the lining epithelium. In the latter variety the tumor is a distinct, circumscribed encapsulated mass of connective tissue.

MYXOMA.

Occurs in sub[illegible] and submucous tissues (in the latter as polypi), and as [illegible]derative process in the connective tissue tumors.

MICROSCOPICAL EXAMINAT ON : — Stellate, branching or spindle-shaped, nucleated cells, lying in a matrix of homogeneous gelatinous material (mucin). Around the cell nuclei there is a large amount of granular protoplasm. From these branching cells there are delicate fibrils given off, which, joining with others, of like origin, form a delicate network of interlacing fibrils. The tissue is poor in blood vessels.

MACROSCOPICAL EXAMINATION :—Tumor generally lobulated with thin fibrous capsule from which thin fibrous trabeculae run into tumor mass. Between these bands the tissue is soft, gelatinous, and, in some cases, of same consistence as mucus.

LIPOMA OR FATTY TUMOR.

Occurs wherever fat is normally found, especially in subcutaneous tissues.

MICROSCOPICAL EXAMINATION :—The fat has the same appearance as that of subcutaneous fat, but the cells are very much larger in size. The trabeculae are rich in blood vessels.

MACROSCOPICAL EXAMINATION :—The tumor is firm, circumscribed, encapsulated, lobulated, possibly fluctuating. On section, tissue is fatty, with greyish glistening fibrous trabeculae.

CHONDROMA OR CARTILAGINOUS TUMOR.

Grows from periosteum of bones, in bones and cartilage, parotid and salivary glands, testicles, lung, skin and breast.

MICROSCOPICAL EXAMINATION :—Has the characters of normal hyaline cartilage.

MACROSCOPIC EXAMINATION : — Usually multiple growths, rounded, lobulated, firm, elastic, cartilaginous and encapsulated.

CHONDROMATA are independent cartilaginous tumors growing where no cartilage is usually found, whereas "ecchondromata" or "enchondromata" are cartilaginous outgrowths from preexisting cartilage.

OSTEOMA OR OSSEOUS TUMOR.

Occurs as outgrowth from bone (exostosis or endostosis), and as a true bony growth. They spring from bone

or cartilage, or their neighbouring connective tissue; in serous membranes and in organs, e.g., testicle and parotid gland.

MICROSCOPICALLY and MACROSCOPICALLY they resemble bone tissue either of the spongy or compact type.

MYOMA OR MUSCLE TUMOR.

Occurs wherever muscular tissue is present, e.g., in uterus. Two forms may be distinguished according as muscle is non-striped (Leiomyoma) or striped (Rhabdomyoma) in character.

MICROSCOPICALLY these have the same characters as muscle tissue.

MACROSCOPICALLY, the tumor is firm, fleshy, elastic, slightly paler than healthy muscle, circumscribed, encapsulated, with connective tissue trabeculae.

NEUROMA OR NERVE TUMOR.

Occurs as medullated nerve fibres (at ends of nerves), or as ganglion cells.

LYMPHANGIOMA OR LYMPHATIC TUMOR.

Due either to an occlusion of a lymphatic vessel with dilatation of its distal areas, or to an actual overgrowth of lymphatic channels. Macroglossia (tongue) and macrocheilia (lip) are good examples of the former type. In these conditions there is obstruction to free outflow of lymph from organ leading to cystic dilatation of pre-existing lymph channels, flattening of their lining endothelial cells, pressure upon surrounding tissues leading to their atrophy and replacement by connective tissue.

HAEMANGIOMA OR BLOOD TUMOR.

Two varieties are distinguished, viz.:

(a). CAVERNOUS HAEMANGIOMA:—Marked dilatation of pre-existing vascular channels which are filled with blood (recognized by the shape and character of the red blood cells). These may be seen in the skin and viscera, especially in the liver. The same changes take place in the intervascular tissue as in lymphangioma.

(b). TELEANGIECTATIC OR CAPILLARY HAEMANGIOMA :—Here the bulk of tumor is formed by an actual overgrowth of blood capillaries. These may be seen as masses of arterioles with thick muscular walls, or as simple tubes with endothelial lining. The endothelial overgrowth may become so marked as to suggest an endothelioma.

MACROSCOPICALLY these are seen as "birth marks" on the skin, either of a deep red (when due to arterial dilatation or overgrowth), or port wine (when due to venous dilatation) color. The capillary form does not project beyond the surface of the skin, and the color does not disappear on pressure.

The cavernous form projects, as a tumor, beyond the surface; except in the liver, in which latter position it is distinguished from a subcapsular haemorrhage by cutting into it, when it is found to extend a short distance into parenchyma of organ.

EPITHELIAL NEW GROWTHS.

These tumors are derived from the epiblastic and hypoblastic germ layers. The simplest form is that of non-malignant epithelioma where there is a simple hyperplasia of epithelial cells especially of the skin. These appear to be well circumscribed and their base is formed by connective tissue. They may form growths much resembling the fibrous papillomata. These epithelial cells do not infiltrate the deeper structures.

ADENOMA OR GLAND TUMOR.

These are benign tumors which spring from glands.

MICROSCOPIC EXAMINATION :—There is a marked proliferation of the epithelial cells lining the glands, and with this overgrowth there is a sprouting, as it were, from the glands, penetrating between the connective tissue bands which tissue may also proliferate. These new glands may either take on the form of those from which they originate, or they may become somewhat irregular in form. The epithelial cells, however, are seen to be placed on a basement membrane which they never penetrate, except when becoming malignant, although they may form irregular masses within the gland tubes and acini.

MACROSCOPIC EXAMINATION :—On the mucous

surfaces they may form a simple thickening of the mucosa, or papillomatous elevations which are distinctly circumscribed, non-infiltrating and not adherent to surrounding structures. On section, the tissue is moderately firm, whitish or reddish-pink in color; rarely surrounded by a fibrous capsule.

EPITHELIAL CYSTOMATA are a variety of epithelial tumors characterized by the formation of large cystic spaces lined with epithelium and filled with fluid. In the same way the glands in ADENOMATA may undergo dilatation, forming cysts. Into both these varieties of tumors papillary growths or excrescences may project, forming cystoma papilliferum and adenocysto-papilliferum.

In adenoma without cyst formation there may be a projection into the ducts of papillomata (adenoma papilliferum).

MALIGNANT CONNECTIVE TISSUE TUMORS.

LYMPHADENOMA OR LYMPHOSARCOMA.

Is a more or less malignant overgrowth of the lymphatic cellular elements (lymphocytes) in sites where these are normally present, and are frequently associated with Hodgkin's disease. They form simple enlargements of lymph glands over the whole body. The individual glands retain their shape and do not tend to coalesce or invade the surrounding structures. In consistence they are either hard or soft.

Both MICROSCOPICALLY and MACROSCOPICALLY the characters are those of normal lymph gland.

SARCOMATA.

In the study of sarcomata the following points, which are common to all varieties, must be clearly made out :—

1st. That they are composed of embryonic connective tissue.

2nd. That they are not encapsulated or circumscribed, but infiltrate locally, and spread through the medium of the vascular channels.

3rd. That they are very vascular, the vessels being of an embryonic type, i.e., their walls are formed of a single layer of elongated tumor cells. In many sections, especially when rather thick, these vessels appear as dark

bands throughout tissues; in others as parallel rows of deeply staining cells, between which may be made out the red blood cells. On cross section they form simply rounded spaces whose walls are very thin and obscure, and can be made out only with the high power. These thin walls are very apt to become ruptured and produce haemorrhages. As these vessels are very difficult to make out in many sections great care must be exercised in finding them, and once having made them out distinctly, their future study is much simpler.

"SARCOMA"—Small Round-Celled.

Grows wherever connective tissue is present in the body, e.g., subcutaneous tissues, fasciae, periosteum of bone and connective tissue of nerves. Tumor is moderately malignant.

MICROSCOPIC EXAMINATION:—Tumor is composed of masses of small round cells (about the size and having the character of lymphocytes) having little or no fibrous tissue matrix and showing no tendency to become circumscribed or encapsulated, but rather to invade all the surrounding tissues. This mass is very vascular, the vessels being of an embryonic type (already described). Throughout tumor haemorrhagic infiltrations may be made out.

MACROSCOPIC EXAMINATION:—Tumor usually rounded, of pale pink color, soft, pulpy, with reddish or brownish haemorrhagic areas throughout. Non-circumscribed, non-encapsulated, but invading extensively the surrounding structures and thus becoming densely adherent to them.

"SARCOMA"—Large Round-Celled.

Occurs in same position as small round-celled, but principally in submucous tissues, forming pale, firm polypoid masses, especially in posterior nares and pharynx. Not so malignant a form as small-celled and rarely forms metastases.

MICROSCOPICALLY differs from the small round-celled variety only by the fact of the cells being two or three times larger.

"SARCOMA"—Small Spindle-Celled.

Occurs in same situations and is of same character microscopically as the other two varieties, but the cells are more highly developed, i.e., they have become more elongated or spindle-shaped.

MACROSCOPICALLY, tumor is rounded, firm, pale, elastic with slight tendency to infiltrate. Some greyish glistening bands of connective tissue through and around it. It is much less malignant than the round celled varieties.

"SARCOMA"—Large Spindle-Celled.

As the foregoing varieties. This differs from small spindle-celled sarcoma in having much larger spindle or oat-shaped cells which in some cases tend to bifurcate or interlace.

MACROSCOPICALLY, they are of a pink color, are softer, more vascular and haemorrhagic than the small spindle-celled variety.

They grow more rapidly and spread frequently through the lymphatics, and are thus exceptions to the general rule.

"SARCOMA"—Recurrent Fibroma.

Occurs especially on the skin as multiple growths.

MICROSCOPICALLY the cells are much more elongated and interlacing than in preceding varieties, forming in many cases mature connective tissue. The vessels are fewer in number and more highly developed than in sarcomatous growths.

MACROSCOPICALLY they form firm pedunculated masses, varying in size from that of a filbert to tumors measuring half a foot in diameter. On section they are firm and have all the appearances of fibroma.

They do not infiltrate the tissues deeply at point of attachment, but they tend to recur locally when excised. This is one of the least malignant types.

"SARCOMA"Osteoid Sarcoma.

A very malignant form growing primarily from periosteum.

MICROSCOPICALLY the cells are larger than in the

small round-celled form, and they are multinucleated. Between these is a very delicate intercellular substance, which, in places, is impregnated with highly refractile granules of calcareous salts. There is no true bone formation as in osteo-sarcoma, but simply a calcification of the cartilaginoid matrix.

"SARCOMA"—Psammoma or Angiolithic Sarcoma.

Occurs in brain membranes, choroid plexus and pineal gland.

MICROSCOPICALLY—Tumor is a small spindle-celled form. These cells are grouped around small vessels or delicate capillaries, their walls being frequently formed by these cells. The cells may be arranged concentrically around vessels. The calcareous salts occur in rounded masses in concentrically arranged whorls.

Tumor may be fibromatous, gliomatous, sarcomatous, adenomatous carcinomatous, angio-fibromatous or angio-sarcomatous in its histological characters.

"SARCOMA"—Alveolar Sarcoma.

Occurs in true skin, pia mater and bone.

MICROSCOPICALLY it is composed of cells resembling much endothelial or epithelial cells. These form round, or oval alveolar masses resembling those seen in carcinomata. Between these cellular masses is a very vascular tissue with a small amount of connective tissue. The cells at periphery of alveolar spaces are somewhat flattened or elongated, concentrically arranged and intimately connected with the fibrous tissue stroma, thus different from carcinomata. No vessels penetrate between these cells.

"SARCOMA"—Myeloid or Giant-Celled.

Grows either within shaft or epiphyses of bone, or under periosteum, especially of femur, tibia humerus or lower maxilla. They are moderately malignant.

MICROSCOPICALLY the tumor is one of the spindle-celled variety, with, in addition, large or small numbers of giant cells. These giant cells are many times larger than the spindle cells and have the same characters as the os-

teoclasts. The nuclei, which are many, are placed in the central portion of the cell.

MACROSCOPICALLY, tumor is moderately firm and elastic; on handling it seems to crackle like parchment under the fingers, and is firmly adherent to all the surrounding structures. On section, the tumor is of a pinkish or brownish color, marked throughout by haemorrhagic infiltrations. When growing from within bone it bulges out the bone and its surface appears to be covered by a thin layer of bone.

"SARCOMA"—Melanotic.

Grows from choroid coat of eye, iris, skin and pia mater (where this pigment or melanin is normally present). It is one of the most malignant forms of sarcoma and very early forms metastatic growths in the organs, especially the liver.

MICROSCOPICALLY, it belongs to the large spindle-celled variety. Around the nuclei of the large spindle cells there is a deposit of a golden, brownish or black-brown pigment (melanin). This is rarely deposited in the intercellular spaces; haemorrhagic infiltrations also present. It is particularly vascular.

MACROSCOPICALLY tumor is a very rapidly growing, blue-black, soft vascular mass, which on section is pinkish in places, in others of a deep brownish or blue-black color. It infiltrates extensively the surrounding structures, to which it becomes most firmly attached.

"SARCOMA"—Osteo-Sarcoma.

Generally grows in connection with bone. Not a very malignant type.

MICROSCOPICALLY they start as ordinary sarcomata being in part composed of rounded or spindle cells; in part of fibrous tissue bands, which push their way through the mass between these cells. In parts may be seen clear, lightly staining areas of bone formation with lamination, and, in some cases, Haversian canals.

MACROSCOPICALLY:—Somewhat similar to myeloid form, but throughout tumor are bony spicules, principally composed of cancellous bone.

GLIOMA.

Gliomata are met with in the nervous system; especially the brain, spinal cord, cranial nerves, and in the eyeball. They are histologically a hyperplasia of neuroglia cells, and are, therefore, not truly malignant. They injure surrounding tissues by pressure. Clinically they sometimes, suggest malignant growths.

MICROSCOPICALLY neuroglia cells contain round or oval nuclei with little surrounding protoplasm. They have fine protoplasmic extensions from the irregular shaped cells, which interlace freely, forming a delicate reticulum. Some of cells are multinucleated.

MACROSCOPICALLY they occur as rounded or oval masses which at periphery gradually merge into nerve tissue. They are of a grey, reddish or pink color. On section, they are greyish, firm, possibly cystic (from central degeneration and softening).

In order to be properly studied it must be examined within a very short time after death, or after its surgical removal.

MALIGNANT EPITHELIAL TUMORS.

CARCINOMA.

These tumors are characterised by the proliferation of epithelial cells, brought about by a cell division (mitosis). The proliferated epithelial cells form atypical masses of cells in alveolar shape, in clusters, or in columns; they infiltrate very extensively all the neighbouring structures, by pushing their way between the muscular and fibrous tissue bundles, especially through the lymphatic chaunels. They spread and form metastases through the lymph channels (an exception to this mode of extension is in carcinoma of stomach, extending to liver through the portal circulation). The lymph glands in neighbourhood are early involved. The connective tissue stroma never penetrates between these clumps of cells.

EPITHELIOMA OR SQUAMOUS-CELLED CANCER.

Arise from cells of the stratified or squamous epithelium of skin or mucous membranes, especially at points of junction of these, e.g., lips, nose, labia, etc.

MACROSCOPIC EXAMINATION :—The squamous cells, especially the prickle cells, of the inter-papillary areas, proliferate, spread downwards and break through the subcutaneous structures in the form of finger-like processes; later irregularly through the lymphatic channels infiltrating very extensively between the tissues of the part in columns or in nests.

In these masses the cells are often found clumped together concentrically; while later they may become dry and horny, as on the surface of skin, this gives the mass of cells the yellowish poorly staining appearance characteristic of a "cell nest" or "epithelial pearl." These nests are characteristic of epitheliomata.

Around these downward growths of epithelial cells there may or may not be a large number of small round cells forming, as it were, a limiting membrane. These later may develop into connective tissue, and thus cause an overgrowth of the fibrous stroma of tumor.

MACROSCOPIC EXAMINATION : — These form warty, cauliflower-shaped nodular masses, or simple thickenings of the skin or mucous membranes. Their surface may be irregular and ulcerated; the tumor mass is firm with extensive infiltration and hardening of the tissues at its base. On section moderately firm; from cut surface a gruel-like fluid can be scraped with the knife (cancer juice), which may contain these epithelial pearls or cell nests. The neighbouring lymphatic glands are enlarged and firm.

RODENT ULCER.

Occurs most frequently on the upper part of the face.

MICROSCOPICALLY, it is composed of pavement epithelium which is arranged in well defined groups, the peripheral cells being sometimes columnar. It does not infiltrate deeply, but surface of skin ulcerates, then cicatrises. It is a very superficial form of epithelioma.

MACROSCOPICALLY, the ulcer is peculiar in that it spreads at one part while other parts cicatrise and become covered with epithelium. The lymph glands are not involved. Tumor is one of the least malignant forms of epithelioma.

CARCINOMA OR CYLINDRICAL-CELLED CANCER.

Occurs on mucous membranes; particularly of alimentary tract, on the skin and in organs; in fact, it occurs in all positions where gland structures are normally present.

MICROSCOPIC EXAMINATION :—These may begin as simple adenomata with proliferation of the glandular cylindrical epithelium. This growth becomes more and more atypical, the epithelial cells spreading between the tissues of affected part, in alveoli of irregular shapes and sizes. These may form tubular masses of cells between the bands of connective tissue, which, in most cases, is also proliferated. Thus from the typical gland these cells may proliferate so rapidly that there is not the slightest attempt at gland formation but merely irregular masses of epithelial cells of all sizes and shapes.

The relative proportion of epithelial elements to connective tissue forms a basis for the present classification of these carcinomata into the following varieties :—(a). Carcinoma simplex; (b). Scirrhus carcinoma; (c). Medullary carcinoma.

(a). CARCINOMA SIMPLEX :—Occurs in glands, especially of the breast, in the form of hard nodular tumors. Cut surface of a greyish-white color.

MICROSCOPICALLY the epithelial cells have no constant shape or size; there is relatively the same amount of connective tissue stroma as of epithelial cells thus giving to it its name.

(b). SCIRRHUS CARCINOMA :—Occurs in breast, testicle, stomach, kidney and ovary, in the form of hard, tough nodular tumors.

MICROSCOPICALLY, in this variety the epithelial cells are rather small and scanty, but there is a relatively large amount of connective tissue stroma.

(c). Medullary Carcinoma :—Occurs in mucous membranes (especially of stomach), and in glands, as a soft, cauliflower-like growth which is particularly liable to ulcerate and bleed.

MICROSCOPICALLY, here we find solid masses of epithelial cells having no lumen and being surrounded by a thin, very delicate connective tissue stroma.

In very few cases do we get a carcinoma of any one of these types throughout, more frequently they present

one type in one area whereas another area may present quite a different type.

CARCINOMA CYLINDROMA.

Is the name given to a type of carcinoma where a cylindrical hyaline degeneration takes place in the centre of these epithelial masses while the unaffected cells are grouped about these hyaline areas. These very rarely occur on skin, mucous membranes and in the glands.

COLLOID CARCINOMA.

A form of carcinoma in which the cellular elements have undergone a colloid degeneration. The colloid material is deposited in the cells in droplets which later coalesce, replacing the whole cell protoplasm. From the deposit of a large amount of this material the alveolar spaces frequently become globular or rounded. This material is quite clear, homogeneous and transparent. Some unaffected or only partially affected cells may be seen in this clear mass. The interalveolar connective tissue is unaffected and distinct.

It occurs in form of firm, gelatinous tumor in the intestines, breast and ovary.

CARCINOMA MYXOMATODES.

Is a form in which the stroma undergoes myxomatous degeneration. It occurs in the intestines and breast.

GIANT-CELLED CARCINOMA.

The cells become particularly large and may be multinucleated. This may be due to hypertrophy, or to mucoid or dropsical degeneration of cells.

MELANO-CARCINOMA.

The pigment, as in melano-sarcoma, lies partly in stroma, and partly in cancer cells. They are rarer than the melano-sarcomata and form gray, brown or black tumors.
